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Foreign Animal Disease Report

United States
Department of Agriculture
Animal and Plant
Health Inspection Service
Veterinary Services

Emergency
Programs



Number 14-1

MAR 27 '86

Spring 1986

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Current Events

Avian Influenza in Pennsylvania

Avian influenza virus H5N2 has been detected in eleven flocks of chickens and one with turkeys located in Snyder, Schuylkill, Fulton, and Northumberland Counties, Pennsylvania, one small broiler flock in Bristol County, Massachusetts, three flocks in Monmouth County, New Jersey, and a broiler flock on Long Island, New York. This is the same antigenic subtype of the virus that resulted in the destruction of more than 17 million chickens, turkeys, and other poultry in southeastern Pennsylvania, Virginia, Maryland, and New Jersey during 1983-84. However, laboratory tests of the virus isolated in January 1986 showed it to be not highly pathogenic. (See 12-2:5.) State agricultural officials quarantined the premises and destroyed the affected chickens and turkeys. State and Federal officials were continuing epidemiological tracing at the time this report was sent to the printer. Preliminary studies suggest that the movement of contaminated crates and trucks associated with marketing live poultry was the means for the introduction of the virus. (Adapted from APHIS Information Service news release.)

Emergency Disease Investigations, 1985

There were 480 diagnostic investigations for possible foreign animal diseases in the 50 States and Puerto Rico during fiscal year (FY) 1985 (October 1, 1984, through September 30, 1985). Of these, 440 cases were reported to the United States Department of Agriculture because of suspected vesicular conditions in cattle, horses, sheep, goats, and swine. Four hundred and thirteen of the investigations were made in the last quarter of the FY as a result of the recurrence of New Jersey type vesicular stomatitis (NJVS) in New Mexico, Arizona, and Colorado. (See 13-4.) Laboratory studies included the testing of serums from animals located in different areas to determine the geographic distribution of NJVS in Colorado and New Mexico. Possible insect vectors and affected livestock were also studied in cooperation with the National Centers for Disease Control (NCDC) and the

Agricultural Research Service (ARS). Results of the studies were not available at the time this report was prepared.

A stallion imported from West Germany was found to be infected with contagious equine metritis (CEM) during May 1985 while being held in quarantine. A great deal of treatment effort was necessary to rid him of the disease. The quarantine was then released. The fractious nature of the animal and a localized secondary infection of the prepuce complicated the treatment.

In October 1984, a pet bird shipped from California to Hawaii was found to be infected with velogenic viscerotropic Newcastle disease (VVND). Surveillance tracing failed to detect further infection. Pet birds were also found to be infected with VVND on three Florida premises from January 17 to February 26, 1985. Two of the positive premises were commercial wholesale facilities. The third was a private aviary. Tracing of sales from the positive premises disclosed an additional case in Puerto Rico. Tracing of birds from the Puerto Rican premises and from the wholesale dealer in Florida to 25 additional States failed to disclose additional cases.

In May 1985, a newly purchased pet bird in a private home in North Carolina was found to be infected with VVND. The virus was also found at the bird's source in Missouri. (Dr. Arthur E. Hall, 301 436-8073.)

Puerto Rico
Tick Program
Update

Specialized training was given to the State and Federal personnel who recently conducted a tick surveillance effort in eastern Puerto Rico. Infested premises were identified and treatment started to eliminate ticks. Surveillance data will be computerized as a first step in the automation of all tick program data. A technical review is scheduled for March 1986.

Tick problems in Puerto Rico are complicated by bovine babesiosis (cattle tick fever). (See 14-4.) By January 23, 1986, a total of 314 premises with approximately 26,676 cattle were under quarantine for babesiosis or exposure to vector ticks. Fifty-four of the herds were diagnosed as infected with babesia based in part upon the results of laboratory examinations of stained blood smears.

Pesticide treatment to destroy ticks in infected herds is being done at 14-day intervals. (Dr. G. P. Combs, 301 436-8097.)

FMD Surveillance
in Mexico

Surveillance activities of the Mexican-American Commission for the Prevention of Foot-and-Mouth Disease (CPA) during 1985 on a total of 119 reports of vesicular diseases in Mexico led to the diagnosis of New Jersey type vesicular stomatitis (NJVS) in 49 herds and Indiana type vesicular stomatitis (INVS) in four herds.

While VS was seen mainly in bovines, in three of the affected herds, horses also had VS. Horses were not affected on 25 premises with bovine VS. On 12 premises with bovine NJVS, where pigs were present, none of the pigs were affected by VS. In 1984, the investigation of 185 reported occurrences of vesicular diseases disclosed NJVS on 94 premises and INVS on one premises. (See 13-1.)

The largest number of affected herds was found in enzootic areas of Veracruz and Guerrero. Foci of affected herds were seen in Tierra Blanca, Veracruz, and in Ometepe and Ayutla, Guerrero. For the first time, NJVS was found in Baja California Sur, in the Municipio of Mulege.

The average attack rate for NJVS in affected bovine herds for lesion cases was 8.0 percent, compared with 6.1 percent in 1984. The attack rate for INVS was 11.5 percent. Attack rates in individual herds ranged from 1 percent to 100 percent. Although one death reportedly was associated with NJVS, it would be difficult to confirm that this death was due to VS.

In only 3 of the total 49 herds investigated was there any introduction of cattle during the month before onset of NJVS. In the remaining 46 there was no history of the introduction of cattle to explain the occurrence of VS.

Most of the occurrences of NJVS were in herds in which the disease had not been seen previously. Owners of 7 of 37 herds with NJVS reported having similar cases before 1985. Also, information obtained during owner interviews suggested that there were large numbers of unreported cases in the vicinity of herds known to be infected.

Of the total 50 affected bovine herds, VS lesions were reported in the mouth in 70 percent, in both mouth and on teats in 20 percent, on teats only in 6 percent, and in both the mouth and on feet in 4 percent. Lesions on the feet only were not reported. (Adapted from the 1985 report of the Mexico-United States Commission for the Prevention of Foot-and-Mouth Disease.)

AI Seminar in Mexico

The Mexico-United States Commission for the Prevention of Foot-and-Mouth Disease (CPA) and the United Nations Food and Agriculture Organization (FAO) conducted a very successful seminar and test exercise for avian influenza (AI) November 23-30, 1985, in Tehuacan, Mexico. Fifty-nine animal disease specialists from Mexico and 16 countries in Central and South America participated. Objectives of the event were to improve the Mexican animal disease emergency response system, evaluate the country's emergency plan for AI, use the training technology developed by CPA, transfer up-to-date information on AI, and exchange national emergency plans. Two days were spent in discussing AI and means to control and eradicate it if it does enter a free area such as Mexico. The remaining 3 days were dedicated to the simulation of an AI outbreak in an imaginary county, to give the participants the experience of dealing with such an emergency. (Adapted from reports received from Dr. John Mason, Mexico-United States Commission for the Prevention of Foot-and-Mouth Disease, 905 531-7600.)

FAD Training Program

November 1985 marked the first foreign animal disease course held at Plum Island for teachers of foreign animal disease courses at colleges of veterinary medicine. Eighteen professors, representing 16 veterinary schools, participated.

Two seminars were planned for FAD diagnosticians. The first was in Lincoln, Nebraska, during the first week in February 1986. It was attended by approximately 55 diagnosticians from the VS Western and Central regions. The second seminar will include approximately 50 diagnosticians from the VS Northern and Southeastern regions and International Operations. The second seminar will be held during May in Hyattsville, Maryland.

Two foreign animal disease schools are planned. The first will be during April, the second in September.

A wildlife disease seminar for FAD diagnosticians is scheduled at the University of Georgia during late May. (Dr. Wesley H. Garnett, 301 436-8091.)

World Animal Disease Roundup

The year 1985 ended with few new developments as far as internationally important animal diseases are concerned. The **foot-and-mouth disease** (FMD) situation in Italy suffered a setback with the occurrence of virus Type C in swine during November. All previous cases in the Italian FMD epidemic were caused by virus Type A. No other cases of FMD were reported from Europe in 1985. However, Europeans keep a close watch on Turkey, troubled by outbreaks caused by viruses Type O₁ and A₂₂. There is also apprehension because of the threat of Type Asia₁, reported lately from Iraq and Malaysia. Elsewhere in Asia, FMD was reported from Hong Kong, Thailand, the Philippines, Oman, and Israel. In the Americas, FMD was reported from Argentina, Brazil, Colombia, Ecuador, Peru, and Venezuela. Numbers of cases in South America are presently decreasing. Chile has reported no FMD for over 18 months.

Swine vesicular disease (SVD) has not been reported since a case in Germany in October 1985. (See 13-4.) However, recent reports indicate the Commonwealth Agricultural Bureaux Laboratory at Pirbright, England, isolated SVD virus from material submitted from Hong Kong in August 1985. **African swine fever** and **hog cholera** (HC) remained relatively static toward the end of 1985, but there was a slight decrease in the number of HC cases reported. Whether this is a beginning payoff for European eradication efforts remains to be seen.

Also of interest are reports on some conditions not heard from for awhile: A case of **Rift Valley fever** was reported in South Africa, a case of **African horse sickness** was reported in Botswana and one in Zimbabwe, and several cases of **lumpy skin disease** were reported in Kenya. (Dr. Hans J. Seyffert, 301 436-8285.)

Focus on...

245 (Maedi and Related Diseases of Sheep //

Maedi initially was described as a slow progressive interstitial pneumonitis of sheep in Iceland and at that time was thought to be a separate distinct entity (G. Gislason, 1947). A chronic encephalitis with severe wasting was described later in Icelandic sheep and called visna (B. Sigurdsson, P. A. Palsson, and H. Grimsson, 1957).

The two are now known to be different manifestations of a common viral infection and are referred to as maedi-visna. This name is used throughout most of the world for similar diseases.

Although maedi-visna has not been reported in the United States, a similar disease has been called progressive pneumonia (H. Marsh, 1923 and 1966), with various modifiers such as Marsh's Montana, chronic, and ovine (E. V. Cowdry and H. Marsh, 1927; C. T. Creech and W. S. Gochemour, 1936; R. C. Cutlip, T. A. Jackson, and H. D. Lehmkuhl, 1979). In this review, we use ovine progressive pneumonia (OPP) to refer to this disease and briefly discuss the history, cause, transmission, susceptible species, clinical signs, lesions, pathogenesis, diagnosis, serology, prevalence, treatment, and control of maedi-visna, OPP, and their causal viruses.

Maedi-visna and OPP are caused by closely related retroviruses that induce severe and progressive emaciation, dyspnea, paralysis, lameness, and indurated udders related to lymphoproliferative and degenerative processes. Sheep that are infected with either virus are carriers for life, even in the presence of high levels of circulating antibodies. Most animals never show evidence of disease. Even though the diseases are similar in most respects, the etiologic viruses differ slightly in nucleic acid sequence. The significance of this difference is unknown, but may represent only strain and not species differences. The two diseases can be discussed together because of the close similarity of the causal agents, clinical signs, and lesions.

History

The respiratory form of OPP was first described in the United States in 1923 in sheep from Montana and was called progressive pneumonia. A report from South Africa in 1915 described a similar disease, later called Graaff-Rinet disease after the region where it was found (D. T. Mitchell, 1915). The diseases that were described in the United States and South Africa were compared and considered identical (E. V. Cowdry and H. Marsh, 1927). Apparently, some sheep had both OPP and pulmonary adenomatosis (jaagsiekte) that led to lingering confusion about the two diseases. We now know that they are distinct entities: **Maedi-visna and OPP are lymphoproliferative and degenerative diseases, and adenomatosis is a carcinomatous disease.**

Maedi-visna was described in France in 1940 and called bouhite, a local name for pulmonary lymphoma, and in Holland in 1943 as zweegerziekte, meaning lagging sickness (F. Lucam, 1942; H. Koens, 1943). In Iceland, the pulmonary form of the disease was described in 1947 under the name maedi, meaning dyspnea, and the paralytic form of the disease was described in 1957 under the name visna, meaning wasting. The diseases were eradicated from Iceland by an extensive slaughter program. They were later shown to be caused by the same virus.

Causes

Maedi-visna and OPP are caused by nononcogenic, exogenous retroviruses of the subfamily Lentivirinae (F. Fenner, 1976). The viruses are closely related in morphological, chemical and serological properties; (L. B. Stone, K. K. Takemoto, and

M. A. Martin, 1971; M. J. Weiss et al., 1977); however, minor differences in nucleic acid sequence have been shown with nucleic acid hybridization techniques (M. J. Weiss et al., 1976; S. M. Roberson, 1982). There is also morphologic similarity and some nucleic acid homology with the viruses of caprine arthritis-encephalitis (CAE) of goats (J. E. Dahlberg, J. E. Gaskin, and K. Perk, 1981) and acquired immune deficiency syndrome (AIDS) of man. (I. M. Chiu et al., 1985; M. A. Gonda et al., 1985; P. Sonigo, 1985). The CAE virus also has close antigenic similarity with maedi-visna and OPP (J. E. Dahlberg, J. M. Gaskin, and K. Perk, 1981). However, humans are not known to be susceptible to the viruses of CAE, maedi-visna, or OPP.

Even though there is morphologic similarity, there is no antigenic relationship with the oncogenic retroviruses (D. H. Harter et al., 1973; L. Stowring, A. T. Haase, and H. P. Charman, 1979). Maedi-visna and OPP viruses consistently cause syncytial formation in cell culture (B. Sigurdsson, P. A. Palsson, and H. Grimsson, 1957), but vary in their cytolytic properties from strain to strain (personal observation). Permissive replication of the viruses can occur in many cell types from different species (D. H. Harter, K. C. Hsu, and H. M. Rose, 1968; C. Torchio, and R. S. Trowbridge, 1977; R. S. Trowbridge, J. F. Schneider, and R. Haddad, 1983). Choroid plexus and fetal lung cells from sheep are most commonly used for virus replication. Virus can either become latent or persistent in cells in culture and in the animal host (V. Barban et al., 1984; A. T. Haase, and H. E. Varmus, 1973). This occurs because the virus forms a provirus; i.e., integration of viral genetic material into that of the host cell. The provirus can lie dormant for an undetermined length of time, probably for life, even in the presence of circulating antibodies. Thus, seropositive sheep are virus carriers.

Transmission

Virus is transmitted to susceptible sheep primarily through the colostrum (G. F. DeBoer, 1970; P. A. Palsson, 1976). Mononuclear cells carrying the provirus in colostrum of infected ewes are probably passed through the intestinal epithelium of the neonate to establish infection. Contact transmission does occur when animals are closely housed, but is less important than milk transmission (B. Sigurdsson, P. A. Palsson, and A. Tryggvadottir, 1953). Transmission in utero can occur, but is rare (R. C. Cutlip, H. D. Lehmkuhl, and T. A. Jackson, 1981).

Susceptible Species

Sheep and goats are the only species known to be susceptible to OPP and maedi-visna (K. L. Banks et al., 1983). Attempts have been made to infect a number of laboratory and other domestic animals, but all have failed (P. A. Palsson, 1976; R. C. Cutlip and H. D. Lehmkuhl, in preparation). There is recent evidence of a distinct breed difference in susceptibility to development of disease, but probably not to infection (R. C. Cutlip, H. D. Lehmkuhl, and K. A. Brogden, In Press). Sheep of the Border Leicester breed were shown to be significantly more susceptible to disease than sheep of the Columbia breed. Circumstantial evidence is accumulating that some other breeds of sheep are highly susceptible to development of disease.

Clinical Signs

Signs develop and progress slowly in both diseases until death ensues after several months to a year (R. C. Cutlip, T. A. Jackson, and H. D. Lehmkuhl, 1979; G. F. DeBoer, 1970; P. A. Paulssen, 1976; R. E. Oliver et al., 1981). Progressive emaciation, in spite of a normal appetite, is frequently the earliest sign. Dyspnea, especially during exercise, is a common finding. Lameness with swollen joints, especially the carpal and tarsal joints, and posterior weakness with ascending paralysis is seen less frequently (R. C. Cutlip et al., 1985a). Lameness and weakness are progressive and may last for months to years. A hard, indurated udder with reduced milk production is sometimes associated with infection (R. C. Cutlip et al., 1985b). Most fatally infected sheep die from secondary bacterial pneumonia. We emphasize that most sheep which are infected with the viruses of maedi-visna or OPP do not succumb to the clinical disease, but once signs are seen there is a relentless course to death.

Lesions

The basic lesion in all affected tissues is the accumulation of lymphatic tissue that often forms nodules that may contain germinal centers. Lungs are enlarged and firm with multiple grey areas and weigh three to four times normal. Lymphatic tissue accumulates in the pulmonary interstitium around airways and blood and lymphatic vessels. In some cases the alveolar epithelium becomes hyperplastic and there is excess fibromuscular tissue. Lesions within the central nervous system consist of varying degrees of accumulation of lymphocytes in the meninges, choroid plexus, and white matter of the brain and spinal cord. In severe cases, they are accompanied with primary demyelination and necrosis. The subependymal and subpial areas of the brain are most involved. Arthritis commonly affects the appendicular joints, but may involve other joints. The lesions are extensive proliferation of synovial and bursal membranes, fibrosis of the joint capsule, and degeneration of articular cartilage and bone. Affected joints are dry (nonsuppurative) and contain some fibrin. The proliferated synovial membranes are hyperemic and infiltrated with lymphocytes, plasmacytes, and macrophages. Lymphocytic mastitis, with multiple accumulations of lymphocytes and plasmacytes in the interstitium and breaks in ductal and acinar epithelium, is a common feature. Fibrinoid necrosis in the tunica media and mononuclear cell accumulations in the tunica adventitia and tunica media are occasionally seen in small muscular arteries of joint capsules, kidneys, brain, and other tissues.

Pathogenesis

Pathogenesis of maedi-visna and OP is best explained as a result of chronic antigenic stimulation related to persistent viral infection. Immunization of infected sheep with visna virus was shown to hasten the development of lesions and, conversely, immunosuppression of infected sheep was shown to retard the development of lesions (N. Nathanson et al., 1981 and 1976). Persistence of the viruses is related to integration of viral nucleic acid into host cell nucleic acid (provirus formation) (A. T. Haase and H. E. Varmus, 1973). The provirus has recently been shown to reside in mononuclear macrophage-type cells of the lungs and in glial cells in the brain (O. Narayan et al., 1982; L. Stowring et al., 1985). Antigenic drift of the virus may also contribute to the persistence, but is less important than integration of viral nucleic acid into host cell nucleic acid (M.

Gutnadottir, 1974; O. Narayan, D. E. Griffin, and J. Chase, 1977; R. Lutley et al., 1983).

Diagnosis

Because of the persistence of virus in the presence of circulating antibodies, a diagnosis of infection by maedi-visna or OPP virus can be made by identifying either virus or specific antibody in the blood. A tentative diagnosis of disease is made from clinical signs and either identification of serum antibodies or isolation of virus. Definitive diagnosis is dependent upon finding characteristic lesions at necropsy in conjunction with clinical signs and demonstration of virus by either serology or isolation.

Serology

Currently, three serologic tests are used to detect antibodies to maedi-visna and OPP viruses: An agar gel immunodiffusion test (AGIDT), enzyme linked immunosorbency assay (ELISA), and indirect immunofluorescent test (IIFT) (R. C. Cutlip, T. A. Jackson, and G. A. Laird, 1977; D. J. Houwers, and A. L. J. Gielkens, 1979; P. Bront, G. Charlier, and A. DeSmet, 1981). The three tests vary only slightly in sensitivity and specificity, but the simplicity of the AGIDT makes it the test of choice for most surveillance or control programs (M. Dawson, 1982). Actively acquired precipitating antibodies to maedi-visna and OPP viruses are slow to develop and with one known exception are maintained for the life of the animal (G. F. DeBoer, 1970; R. Lutley et al., 1983; L. Sihvonen, T. Estola, and J. Tuomi, 1980). Thus, actively acquired antibodies indicate that the animal is infected with virus. Passively acquired antibodies are obtained through the colostrum and are lost by 6 months of age. Therefore, serologic tests are uninterpretable during the first 6 months of life. During this time, a positive test indicates that the animal is either infected and has produced antibodies or has passive antibodies; a negative test indicates that the animal is either noninfected or is infected and has not yet produced antibodies. After 6 months of age, a positive test indicates that the animal is persistently infected; a negative test is interpreted the same as one before 6 months of age. Some seropositive sheep or goats will become seronegative following parturition because of the large quantity of antibodies lost in the colostrum. Because of these possibilities, serologic testing is best done on a flock or herd basis.

Prevalence

Prevalence of infection and of disease vary greatly. Retroviruses of sheep are reported worldwide except in Iceland, Australia, and New Zealand. Ovine progressive pneumonia has been reported in the United States and Israel (R. C. Cutlip, T. A. Jackson, and G. A. Laird, 1977; N. L. Gates et al., 1978; E. M. Huffman et al., 1978; E. M. Huffman et al., 1981). In the United States, as many as 50 percent of the total sheep population may be infected. (R. C. Cutlip, T. A. Jackson, and G. A. Laird, 1977; N. L. Gates et al., 1978; E. M. Huffman et al., 1981). Within individual flocks, infection increases with age. This may indicate lateral transmission of virus or delayed seroconversion after infection at birth. The percent of infected sheep that develop clinical disease is not known, but as stated, certain breeds of sheep are more susceptible to disease than others (R. C. Cutlip, H. D. Lehmkuhl, and K. A. Brogden, in press).

Treatment	No effective treatment for the viral infection is known, so treatment must be symptomatic. Anti-inflammatory drugs can be used to reduce joint pain and antibiotics can be used to prevent secondary bacterial infections, especially of the lungs.
Control	Vaccines against maedi-visna and OPP have not been successful (R. C. Cutlip, <u>et al.</u> , in preparation), but several laboratories, including the Agricultural Research Service laboratory at Ames, Iowa, have eradicated maedi-visna and OPP viruses from experimental sheep by collecting offspring at birth and rearing them in isolation either on pasteurized milk or a milk substitute (M. R. Light <u>et al.</u> , 1979; D. J. Houwers <u>et al.</u> , 1983). We and others have also eradicated the viruses from experimental sheep by a test and removal procedure (D. J. Houwers, J. Schaake, Jr., and G. F. DeBoer, 1984; R. C. Cutlip and H. D. Lehmkuhl, In Press). We recommend the following two methods of control:

Method 1. Test and remove.

1. Bleed all sheep and goats in the flock and test serologically for antibodies to maedi-visna and OPP viruses. All sheep and goats on the farm must be included because both viruses can infect either species and cause a positive reading of the serological test.
2. Remove from the flock all seropositive animals and their progeny that are less than 1 year old. Animals removed from the herd can either be sold for slaughter or isolated in separate facilities. Slaughter is recommended because of the danger of cross contamination.
3. Keep the clean flock isolated from infected sheep and goats and from people and equipment in contact with an infected flock.
4. Add only seronegative animals to the flock, either from other seronegative herds or from seronegative parents in an infected flock following 1 year of isolation with a negative test reading.
5. Testing must be done annually until there are at least two consecutive negative flock tests to be reasonably sure that the flock is free of the virus.

Method II. Isolate and artificially rear progeny.

1. Remove progeny from dams before nursing and maintain in isolation.
2. Proceed with steps 3 and 4 described in test and removal method above to assure virus free status of the new herd. Annual serologic tests are recommended.

General
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on Maedi-Visna
and OPP

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